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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
10/523,588	02/04/2005	Helen Francis-Lang	05-940-F (EX03-057C-US)	4379
63572 7590 10/06/2009 MCDONNELL BOEHNEN HULBERT @ BERGHOFF LLP 300 SOUTH WACKER DRIVE SUITE 3100 CHICAGO, IL 60606			EXAMINER SWOPE, SHERIDAN	
			ART UNIT 1652	PAPER NUMBER
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**Please find below and/or attached an Office communication concerning this application or proceeding.**

The time period for reply, if any, is set in the attached communication.

<b>Office Action Summary</b>	<b>Application No.</b> 10/523,588	<b>Applicant(s)</b> FRANCIS-LANG ET AL.	
	<b>Examiner</b> SHERIDAN SWOPE	<b>Art Unit</b> 1652	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

### Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

### Status

- 1) ☒ Responsive to communication(s) filed on 30 June 2009.
- 2a) ☒ This action is **FINAL**.                      2b) ☐ This action is non-final.
- 3) ☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

### Disposition of Claims

- 4) ☒ Claim(s) 1,3-5,7-11,13,15,16,20 and 22-30 is/are pending in the application.
- 4a) Of the above claim(s) 4,5,7-11,13,15,20 and 22-25 is/are withdrawn from consideration.
- 5) ☐ Claim(s) \_\_\_\_\_ is/are allowed.
- 6) ☒ Claim(s) 1,3,16 and 26-30 is/are rejected.
- 7) ☐ Claim(s) \_\_\_\_\_ is/are objected to.
- 8) ☐ Claim(s) \_\_\_\_\_ are subject to restriction and/or election requirement.

### Application Papers

- 9) ☒ The specification is objected to by the Examiner.
- 10) ☐ The drawing(s) filed on \_\_\_\_\_ is/are: a) ☐ accepted or b) ☐ objected to by the Examiner.  
Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).  
Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).
- 11) ☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

### Priority under 35 U.S.C. § 119

- 12) ☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).
- a) ☐ All    b) ☐ Some \*    c) ☐ None of:
1. ☐ Certified copies of the priority documents have been received.
  2. ☐ Certified copies of the priority documents have been received in Application No. \_\_\_\_\_.
  3. ☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

\* See the attached detailed Office action for a list of the certified copies not received.

### Attachment(s)

- |  |   |
|--|---|
| 1) <input checked="" type="checkbox"/> Notice of References Cited (PTO-892)          | 4) <input type="checkbox"/> Interview Summary (PTO-413)           |
| 2) <input type="checkbox"/> Notice of Draftsperson's Patent Drawing Review (PTO-948) | Paper No(s)/Mail Date. _____                                      |
| 3) <input type="checkbox"/> Information Disclosure Statement(s) (PTO/SB/08)          | 5) <input type="checkbox"/> Notice of Informal Patent Application |
| Paper No(s)/Mail Date _____  | 6) <input type="checkbox"/> Other: _____                          |

**DETAILED ACTION**

Applicants' response of June 30, 2009, to the action of December 20, 2008, is acknowledged. It is acknowledged that Claim 6 has been cancelled, Claims 1 and 16 have been amended, and Claims 26-30 have been added. Claims 1, 3-5, 7-11, 13, 15, 16, 20, and 22-30 are pending. The elected invention is directed to a method for identifying a p21 pathway modulator using a cellular proliferation assay system comprising a casein kinase $\gamma$ . Claims 4, 5, 7-11, 13, 15, 20, and 22-25 were previously withdrawn from further consideration pursuant to 37 CFR 1.142(b) as being drawn to nonelected inventions. Claims 1, 3, 16, and 26-30 are hereby examined.

It is noted that Applicants state in their remarks (pg 6, parag 4) that New Claims 26-30 are drawn to subject matter found in original Claims 7, 8, 9, 10, and 11, which represent Inventions II-VII. However, New Claims 26-30 are not drawn to subject matter found in original claims 7, 8, 9, 10, and 11.

***Specification-Objections***

The specification is objected to because "Eraser" [0117] should be "Fraser".

The specification is objected to for referencing Bourne et al, 1990 and Marshall et al, 1991 [0010, 0117] when referring to p21. The protein of Bourne et al and Marshall et al is the p21ras GTPase, which is a structurally and functionally different protein from the cyclin dependent kinase inhibitor, p21CDKN1/WAF1/CIP1, discussed in the instant application.

The specification is objected to for referencing Hay et al 1994 when referring to the GMR-p21 transgene [0117]. Hay et al fails to comprise the term p21, GMR-p21, or GMR-p21 transgene.

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***Claim Rejections - 35 USC § 101***

35 U.S.C. 101 reads as follows:

Whoever invents or discovers any new and useful process, machine, manufacture, or composition of matter, or any new and useful improvement thereof, may obtain a patent therefor, subject to the conditions and requirements of this title.

***Utility***

Rejection of Claims 1 and 3 under 35 U.S.C. 101 because the claimed invention lacks patentable utility, as explained in the prior action, is maintained. Claims 26-30 are herein rejected under 35 U.S.C. 101 for the same reasons.

In support of their request that said rejection be withdrawn, Applicants provide the following arguments. An invention has specific utility if the identified use is well-defined and has a particular benefit to the public and is specific to the subject matter claimed. Agents identified using the recited method can be used in the study and treatment of disorders associated with defective or impaired p21 function, such as cancer. (specification; pg 2-4). Contrary to the Office's assertion, the claimed invention does indeed have an immediate benefit to the public because it defines a "real world" or "practical" use.

These arguments are not found to be persuasive for the following reasons. It is acknowledged that an invention has specific utility if the identified use is well-defined and has a particular benefit to the public and is specific to the subject matter claimed. However, as acknowledged by Applicants, the use for compounds identified with the recited method is for further investigation. Thus, without additional experimentation, any compound identified by the recited method cannot be used for study or treatment of disorders associated with defective p21 function. The recited method is not substantial because a use for any identified compounds is only potential and not in currently available in practical form.

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See MPEP 2107.01 IB(C) which states:

“the following are examples of situations that require or constitute carrying out further research to identify or reasonably confirm a "real world" context of use and, therefore, do not define "substantial utilities":

(C) A method of assaying for or identifying a material that itself has no specific and/or substantial utility;”

Further rejection of Claims 1 and 3 under 35 U.S.C. 112, first paragraph, for the reasons explained in the prior action, is maintained. Claims 26-30 are herein also rejected under 35 U.S.C. 112, first paragraph, for the same reasons.

***Claim Rejections - 35 USC § 112-Second Paragraph***

The following is a quotation of the second paragraph of 35 U.S.C. 112:

The specification shall conclude with one or more claims particularly pointing out and distinctly claiming the subject matter, which the applicant regards as his invention.

Claims 1, 3, 16, and 26-30 are rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention for the following reason.

For Claims 1 and 16, the phrase “full-length wildtype CSNK1G polypeptides and nucleic acids” renders the claims indefinite. The specification states:

“The term “CSNK1G polypeptide” refers to a full-length CSNK1G protein or a functionally active fragment or derivative thereof. A “functionally active” CSNK1G fragment or derivative exhibits one or more functional activities associated with a full-length, wild-type CSNK1G protein, such as antigenic or immunogenic activity, enzymatic activity, ability to bind natural cellular substrates, etc.” (pg 4)

Said definition is only exemplary and does not provide any structural or functional limitations for the genus of any “full-length wildtype CSNK1G polypeptides and nucleic acids”. The skilled artisan would not know the metes and bounds of the recited invention. In addition, the art teaches there are several isoforms of CSNK1G (Zhai et al, 1995; IDS); the specification

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fails to define which isoforms of which organisms/microorganisms are encompassed. Claims 3 and 26-30, as dependent from Claim 1 are indefinite for the same reasons.

For Claims 1 and 16, the term “wildtype” renders the claim indefinite. It is unclear whether said term encompasses CSNK1G polypeptides and nucleic acids that are (i) biomolecules expressed only in wild organisms/microorganisms occurring in nature; ie, not domesticated or grown by man, (ii) recombinant biomolecules derived only from wild organisms/microorganisms occurring in nature; ie, not domesticated or grown by man, and/or (iii) endogenous or recombinant biomolecules expressed in organisms/microorganisms that have been grown or altered by man, ie, any domesticated, mutated, or recombinant organisms/microorganisms. The skilled artisan would not know the metes and bounds of the recited invention. Claims 3 and 26-30, as dependent from Claim 1 are indefinite for the same reasons.

For Claim 16(d)(f), the phrase “phenotypic change” renders the claim indefinite. The specification states:

“Preferably, the modulating agent produces a detectable *phenotypic change* in the cell indicating that the p21 function is restored. The phrase “function is restored”, and equivalents, as used herein, means that the desired phenotype is achieved, or is brought closer to normal compared to untreated cells. For example, with restored p21 function, cell proliferation and/or progression through cell cycle may normalize, or be brought closer to normal relative to untreated cells.” [0110]

Said definition is only exemplary and fails to define the metes and bounds of “phenotypic change”. The skilled artisan would not know the metes and bounds of the recited invention.

Claims 3 and 16 are rendered indefinite for improper antecedent usage as follows.

For Claim 3, the phrase “the cultured cells” should be corrected to “the mammalian cultured cells”.

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For Claim 16(f)line1, the phrase “a phenotype change” should be corrected to “the phenotype change”.

Any subsequent rejection based, on clarification of the above phrases and terms, will not be considered a new ground for rejection.

***Claim Rejections - 35 USC § 112-First Paragraph***

The following is a quotation of the first paragraph of 35 U.S.C. 112:

The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

**Enablement**

The rejection of Claims 1, 3, and 16 under 35 U.S.C. 112, first paragraph/enablement, for reasons explained in the prior action, is maintained. Claims 26-30 are herein rejected under 35 U.S.C. 112, first paragraph/enablement, for the same reasons.

In support of their request that said rejection be withdrawn, Applicants provide the following arguments.

(A) The specification at pages 4-5 clearly describes and provides examples of full-length wildtype CSNK1G polypeptides and nucleic acids, including, for example, those CSNK1G molecules having SEQ ID NOs: 1-16. The specification further teaches methods for obtaining additional CSNK1G sequences (pg 7-9).

(B) The specification teaches that the p21 pathway is modulated by CSNK1G, as evidenced by the Drosophila data provided and discussed on pages 1-3 and 34. Applicants' findings clearly show that the CG6963 gene modifies the p21 pathway and that the vertebrate orthologs of the CG6963 gene are the CSNK1G genes. The Drosophila model used to generate

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this data is a well-known research tool and is extensively utilized as a model of human disease, especially for understanding novel genes (Adams et al, 2002). The scientific community's understanding of many human genes is due to research in *Drosophila* (Chintapalli et al, 2007). The Chintapalli reference specifically confirms the usefulness of the *Drosophila* system to identify the function of human genes involved in disease and a variety of pathways that were discovered using the *Drosophila* model.

(C) As taught in the specification and known in the art, the p21 pathway is involved in the regulation of cell growth and proliferation (specification, pg 1; Funk et al, 2000). Moreover, Applicants have demonstrated that CSNK1G is overexpressed in various tumor cell lines (pg 37-38) and have also demonstrated that siRNA against CSNK1G (ie, decreased CSNK1G expression) decreases proliferation in LXI, 231T, and A549 cells (pg 38-39).

(D) The specification teaches that one can use cultured cells or animal models defective in p21 to confirm that the test agent is modulating the p21 pathway and that the p21 pathway is mediating cell proliferation (pg 12, 24-25, and 30).

The specification also teaches that genetically modified animals having altered p21 expression, such as p21 knock-out or p21 knock-in animals, can be used to further assess the role of CSNK1G in a p21 pathway process, such as cell proliferation. (pg 9-12 and 30). This type of testing involves techniques well-known in the art and thus would not be considered undue experimentation for the skilled artisan.

These arguments are not found to be persuasive for the following reasons.

(A) Reply: It is acknowledged that said pages of the specification provide some examples of known human CSNK1G polypeptides and nucleic acids. However, said pages are



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only exemplary and do not provide any structural or functional limitations for the genus of any “full-length wildtype CSNK1G polypeptides and nucleic acids”. Therefore, the skilled artisan is not enabled for making and using said genus.

(B) Reply: The specification, pages 1-3, (i) describe some prior art on molecules p21 and casein kinase I, (ii) describe that *Drosophila* has been used to analyze biochemical processes that, in some cases, are relevant to vertebrates, (iii) assert that Applicants have discovered that CSNK1G modulates the p21 pathway, and (iv) assert that known methods can be used to further study how CSNK1G modulates the p21 pathway. Nowhere do pages 1-3 provide any evidence that CSNK1G acts via the p21 pathway.

The specification, page 34, teaches the following. One set of flies over-expressing p21 were produced and the eyes of these flies had a mutant phenotype, “reduced, rough eyes”. A second set of flies were produced, wherein the flies had random genetic mutations via transposon-mediated mutation. The two set of flies were crossed. It was found that mutation in GISH (*Drosophila* CSNK1G) acted as an “enhancer of the eye phenotype”.

Based on these teachings, the skilled artisan would not concluded that, more likely than not, CSNK1G acts via the p21 pathway for the following reasons. First, it is unclear what the specification means by “enhancer of the eye phenotype”: does this mean that CSNK1G (i) reversed the effect of p21 over-expression to produce a more normal eye or (ii) enhanced the eye degeneration? Second, the specification provides no evidence that p21 and CSNK1G act via the same signal transduction pathways in the development of “rough eye”. Third, the art teaches that “rough eye” in *Drosophila* is a gross phenotype that is affected by many signaling pathways (Kumar et al, 1997). For example, Wolff et al, (1991) teaches that mutation in either the *echinus*

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or *roughest* gene both result in “rough eye” (pg , parag 2-4; Fig 4) and that the mechanism by which “rough eye” develops is distinct for these two mutants (Fig 5-6; pg 834, parag 3). Without further experimentation at the molecular level, the skilled artisan cannot deduce that, more likely than not, any effects of p21 and CSNK1G on “rough eye” are mediated by the same signal transduction pathway.

It is acknowledged that *Drosophila* is a useful model for some mammalian pathways and disorders. However, not all mammalian pathways and disorders can be modeled in *Drosophila*. In fact, very relevant to the instant claims, Ollmann et al, 2000 teaches that *Drosophila* is not a useful model for the role of the mammalian p53/p21 pathway in inducing cell cycle arrest at G1 (parag brdg pg 94-95; pg 97, parag 3; Fig 4E-F). Chintapalli et al cannot be used as evidence for enablement because it was published after this Application's priority date. Moreover, neither Adams nor Chintapalli mention p21, CSNK1G, a CSNK1G/p21 pathway, or any conditions mediated thereby. Searches of the STN, EAST, and NCBI/Entrez databases failed to teach *Drosophila* as a model for a mammalian CSNK1G/p21 pathway or any disorder due to alteration of a CSNK1G/p21 pathway in mammals (search results of record). Based on the fact that the prior art does not teach that any CSNK1G/p21 pathway in *Drosophila* is a model for a CSNK1G/p21 pathway in mammals, the public must look to the specification for said teachings. However, the specification only asserts that *Drosophila* is a model for a CSNK1G/p21 pathway in mammals without providing any evidence for said assertion. Based on said lack of evidence, and in light of the teachings of Ollmann et al, the skilled artisan would not conclude that, more likely than not, a CSNK1G/p21 pathway in *Drosophila* is a model for a CSNK1G/p21 pathway in mammals.

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(C) Reply: It is acknowledged that the p21 pathway is involved in the regulation of cell growth and proliferation in some systems. It is also acknowledged that the specification teaches that SEQ ID NO: 1, 8, and 11 are modestly elevated in some tumor cells and that RNAi of SEQ ID NO: 1, 8, and 11 appears to decrease proliferation in LX1, 231T, A549 cell lines. However, most importantly, the specification fails to provide a link between any CSNK1G-mediated proliferation and any p21-mediated proliferation in any mammalian cell system.

(D) Reply: It is acknowledged that the specification asserts that one can use cultured cells or animal models defective in p21 to confirm that the test agent is modulating the p21 pathway and that the effects of any test agent on cell proliferation are via the p21 pathway. However, the specification fails to provide any evidence for said assertions. Moreover, the prior art teaches that the function of p21 is redundant with other proteins and that p21 knock-out mice develop normally and cannot necessarily be used to determine the effects of p21 activity (Brooks et al, 1998, pg 305, parag 2; Zhang et al, 2009, pg 217, parag 2, pg 219, parag 3).

It is acknowledged that the specification asserts that one can use animal models altered in p21 to assess the role of CSNK1G in p21 pathway processes, such as cell proliferation. (pages 9-12 and 30). However, the specification fails to provide any evidence for said assertion. As explained above, the prior teaches that p21 is functionally redundant with other proteins and that p21 knock-out mice cannot necessarily be used to determine p21-signal transduction pathways. The prior art also teaches that there are a multitude of CSNK1G proteins and polynucleotides having overlapping and distinct functions (Zhai et al, 1995, Fig 6-10; Lussier et al, 1997, Abstract, pg 2693, parag 4; Morgan-Lappe et al, 2006, Fig 4, parag brdg pg 1346-47). The skilled artisan would not conclude that, more likely than not, all CSNK1G proteins act via p21. The

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specification fails to provide any guidance as to which CSNK1G proteins act via p21 in which systems. Testing all CSNK1G proteins for acting via p21 in all cellular and in vivo systems represents undue experimentation.

For these reasons and those explained in the prior actions, Claims 1, 3, 16, and 26-30 are rejected under 35 U.S.C. 112, first paragraph/enablement.

### **Written Description**

Rejection of Claims 1, 3, and 16 under 35 U.S.C. 112, first paragraph/written description, for reasons explained in the prior action, is maintained. Claims 26-30 are herein rejected under 35 U.S.C. 112, first paragraph/written description, for the same reasons.

In support of their request that said rejection be withdrawn, Applicants provide the following arguments.

(A) The claims have been amended to recite a method for identifying a candidate p21 pathway modulator employing a mammalian cell that expresses a full-length, wildtype CSNK1G polypeptide or nucleic acid in a cell proliferation assay system using a test agent that modulates the expression of CSNK1G. The specification sufficiently describes and provides a representative number of exemplary cultured mammalian cells that can be used in the claimed methods.

(B) Applicants reiterate their arguments, set forth above, for the enablement rejection.

These arguments are not found to be persuasive for the following reasons.

(A) Reply: It is acknowledged that the claims have been so amended and that the specification asserts that certain mammalian cells that can be used in the claimed methods. However, the specification fails to provide evidence that any mammalian cell can be used in the

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claimed methods. No example is provided for a method, using an inhibitor of CSNK1G expression, for identifying a p21 pathway modulator using a mammalian cell. Said method was not described in the specification in such a way that the skilled artisan would recognize that Applicants were in possession at the time of filing. Applicants merely rely on the results from *Drosophila* and assert that the recited method can be performed in mammalian cells; leaving to the public the task of determining if said assertion is correct.

(B) These arguments are not found to be persuasive for the reasons explained above.

Claim 26 is rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the written description requirement. Claim 26 contains subject matter that was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the Inventors, at the time the application was filed, had possession of the claimed invention. Claim 26, as dependent on Claim 1, introduces the limitation of contacting a mammalian cell with an anti-CSNK1G antibody that modulates the activity of CSNK1G inside the mammalian cell. The specification fails to describe said limitation and, thus, Claim 26 is rejected under 35 U.S.C. 112, first paragraph, for introducing New Matter.

Claim 30 is rejected under 35 U.S.C. 112, first paragraph, as containing subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention. Claim 30 is directed to a genus of methods, using an agent that modulates CSNK1G activity, to restore the function of p21 in mammalian p21 knock-out cells. The specification fails to describe any successful species of said genus of methods. Given this lack of description of representative species encompassed by the genera of the claims, the

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specification fails to sufficiently describe the claimed invention in such full, clear, concise, and exact terms that a skilled artisan would recognize that applicants were in possession of the claimed invention.

For these reasons and those explained in the prior actions, Claims 1, 3, 16, and 26-30 are rejected under 35 U.S.C. 112, paragraph/written description.

***Allowable Subject Matter***

No claims are allowable.

Applicant's amendment necessitated any new grounds of rejection presented in this Office action. Any new references were cited solely to support rejection(s) based on amendment or rebut Applicants' arguments. Accordingly, **THIS ACTION IS MADE FINAL**. See MPEP § 706.07(a). Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the date of this final action.

Regarding filing an Appeal, Applicants are referred to the Official Gazette Notice published July 12, 2005 describing the Pre-Appeal Brief Review Program.

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### **Final Comments**

To insure that each document is properly filed in the electronic file wrapper, it is requested that each of amendments to the specification, amendments to the claims, Applicants' remarks, requests for extension of time, and any other distinct papers be submitted on separate pages.

It is also requested that Applicants identify support, within the original application, for any amendments to the claims and specification.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Sheridan L. Swope whose telephone number is 571-272-0943. The examiner can normally be reached on M-F; 9:30-7 EST.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Andrew Wang can be reached on 571-272-0811. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published application may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see <http://pair-direct.uspto.gov>. Should you have questions on the access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free).

/SHERIDAN SWOPE/  
Primary Examiner, Art Unit 1652